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Ginkgo biloba extracts ameliorates apoptosis and renal damage in chronic kidney disease rat model

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Objectives: More than one fifth of people over ages of 65 years have some degrees of chronic kidney disease (CKD). Oxidative stress is an important factor contributing to kidney damage by increasing production of oxidants, particularly insufficiency of endogenous antioxidant defense system
Ginkgo biloba extracts (GBE), a phytoestrogen, has been widely used for treating coronary heart disease and neurological disease in clinical settings and demonstrated beneficial effects at biochemical and pharmacological levels.
This study was carried out to examine the effects of GBE in adenine-induced tubular epithelial apoptosis, signaling pathway and renal damage in chronic kidney disease rat model.

Methods: A rat model of renal damage was created by adenine. Rats in Normal and Vehicle groups received distilled water. Rats were given aqueous extract of GBE dose (100, 200 and 500 mg/body weight and allopurinol. Proteinuria; urinary N-acetyl-β-D-glucosaminidase (NAG) levels; the blood biochemical parameters; renal histopathology damage; transferase-mediated dUTP nick-end labeling (TUNEL)-staining; the key molecular protein expressions in mitochondrial and transforming growth factor (TGF)-β1-c-JunNH2-terminal kinase (JNK) pathways were examined, respectively.

Results: Adenine administration induced severe renal damages, as indicated by the mass proteinuria, the heavy urinary NAG, and the marked histopathological injury in tubules and interstitium. This was associated with the activation of TGF-β1-JNK signaling pathway and tubular epithelial apoptosis. GBE treatment, however, significantly prevented proteinuria and urinary NAG elevation, and attenuated tubular epithelial apoptosis in dose dependent manner. GEB (500mg/body) is most effective to normalize renal damage and restoration. It suppressed the protein expressions of Bax and cleaved caspase-3, whereas it enhanced the protein expression of Bcl-2. Furthermore, it also suppressed the protein levels of TGF-β1 as well as phosphorylated-JNK (p-JNK).

Conclusions: This study suggests renoprotective and antioxidant enhancing effects for GBE in a rat model of CKD, presumably through the suppression of TGF-β1-JNK pathway activation..